

| Project Title | Funding | Strategic Plan Objective | Institution |
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| To study the relationship between low GAD2 levels and anti-GAD antibodies in autistic children | \$7,260 | Q2.S.A | Hartwick College |
| The Study of Toddlers with Autism and Regression (STAR) Protocol – Screening for treatable disorders and biomarkers of inflammation and immune activation in the plasma and CNS | \$0 | Q2.S.A | Surrey Place Centre, Toronto |
| The mechanism of the maternal infection risk factor for autism | \$150,000 | Q2.S.A | California Institute of Technology |
| Systematic characterization of the immune response to gluten and casein in autism spectrum disorders | \$0 | Q2.S.A | Weill Cornell Medical College |
| Sensitive periods in cerebellar development | \$32,941 | Q2.S.A | University of Maryland, Baltimore |
| Role of microglial activation in the serotonergic and neuroimmune disturbances underlying autism | \$50,000 | Q2.S.A | Hamamatsu University School of Medicine |
| Role of microglia and complement at developing synapses in ASD | \$60,001 | Q2.S.A | Boston Children's Hospital |
| Redox abnormalities as a vulnerability phenotype for autism and related alterations in CNS development | \$0 | Q2.S.A | State University of New York at Potsdam |
| Redox abnormalities as a vulnerability phenotype for autism and related alterations in CNS development | \$0 | Q2.S.A | Arkansas Children's Hospital Research Institute |
| Redox abnormalities as a vulnerability phenotype for autism and related alterations in CNS development | \$0 | Q2.S.A | University of Rochester |
| Prostaglandins and cerebellum development | \$371,250 | Q2.S.A | University of Maryland, Baltimore |
| Project 2: Immunological susceptibility of autism (supplement) | \$30,784 | Q2.S.A | University of California, Davis |
| Neuroprotective effects of oxytocin receptor signaling in the enteric nervous system | \$25,000 | Q2.Other | Columbia University |
| Neuroimmunologic investigations of autism spectrum disorders (ASD) | \$101,877 | Q2.S.F | National Institutes of Health |
| Mechanisms of synaptic alterations in a neuroinflammation model of autism | \$579,882 | Q2.S.A | University of Nebraska Medical Center |
| Mechanisms of mitochondrial dysfunction in autism | \$0 | Q2.S.A | Georgia State University |
| Influence of maternal cytokines during pregnancy on effector and regulatory T helper cells as etiological factors in autism | \$0 | Q2.S.A | University of Medicine & Dentistry of New Jersey |
| IL-1beta and IL1RAPL1: Gene-environment interactions regulating synapse density and function in ASD | \$28,600 | Q2.S.A | University of California, Davis |
| Hyperthermia and the amelioration of autism symptoms | \$66,153 | Q2.S.A | Montefiore Medical Center |
| GABRB3 and placental vulnerability in ASD | \$642,258 | Q2.S.A | Stanford University |
| GABA(A) and prenatal immune events leading to autism | \$125,000 | Q2.S.A | Stanford University |
| Exploring metabolic dysfunction in the brains of people with autism | \$0 | Q2.S.A | George Washington University |
| Convergence of immune and genetic signaling pathways in autism and schizophrenia | \$0 | Q2.S.A | University of California, Davis |
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| Brain mitochondrial abnormalities in autism | \$20,000 | Q2.S.A | New York State Institute for Basic Research in Developmental Disabilities |
| Autoimmunity against novel antigens in neuropsychiatric dysfunction | \$320,000 | Q2.S.A | University of Pennsylvania |
| Autism spectrum disorders –inflammatory subtype: Molecular characterization | \$30,000 | Q2.S.A | University of Medicine & Dentistry of New Jersey |
| A non-human primate autism model based on maternal infection | \$0 | Q2.S.A | California Institute of Technology |
| Altered placental tryptophan metabolism: A crucial molecular pathway for the fetal programming of neurodevelopmental disorders | \$535,699 | Q2.S.A | University of Southern California |
| 3 Tesla 31Phosphorus magnetic resonance spectroscopy in disorder with abnormal bioenergetics | \$3,250 | Q2.Other | Massachusetts General Hospital |

